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ARTICLES

SEVERE INFANTILE MALNUTRITION AND ITS MANAGEMENT*

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Malnutrition used to be the main pediatric problem all over the world until the early years of this century. This was true in many parts of Europe when these countries had not reached their present stage of social, economic, and cultural development. As a matter of fact, in some areas, malnutrition itself had a large influence on the birth of pediatrics as a separate specialty in the field of medicine. When the general practitioner or the internist realized that his management of severaly malnourished infants often resulted in failure, the need for specialists in the field became vividly apparent, and it then became the job of those physicians who devoted themselves particularly to the problems of infancy and childhood to be responsible for such cases.

Infantile malnutrition is still the main problem in those countries which have the most room for development. Many of the Asian, African and Latin American countries are in the same position, and in these areas malnutrition is the most important single cause of infant mortality. In many cases it will be found that although the immediate cause of death may be recorded as a gastro-intestinal or lower respir-

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atory infection, or one of the childhood exanthemata, the patient also suffered from some degree of malnutrition which was actually the basic, even though not the immediate, reason for the fatal outcome. Hospitals and clinics in these countries are crowded with cases of malnutrition. This has been our experience at the Hacettepe Children's Hospital of Ankara, which began functioning a year ago, in October 1957.

We are extremely interested in the problem of malnutrition and are convinced that weight is the most convenient and perhaps the best single guide to the nutritional state. When nutrition is inadequate, growth slows or stops altogether and weight, plotted againts chronological age, provides a simple, available index of growth. We have taken the Mexican figures as our standards for theoretical average weight.[1] According to these norms, the weight at birth is taken to be 3,200

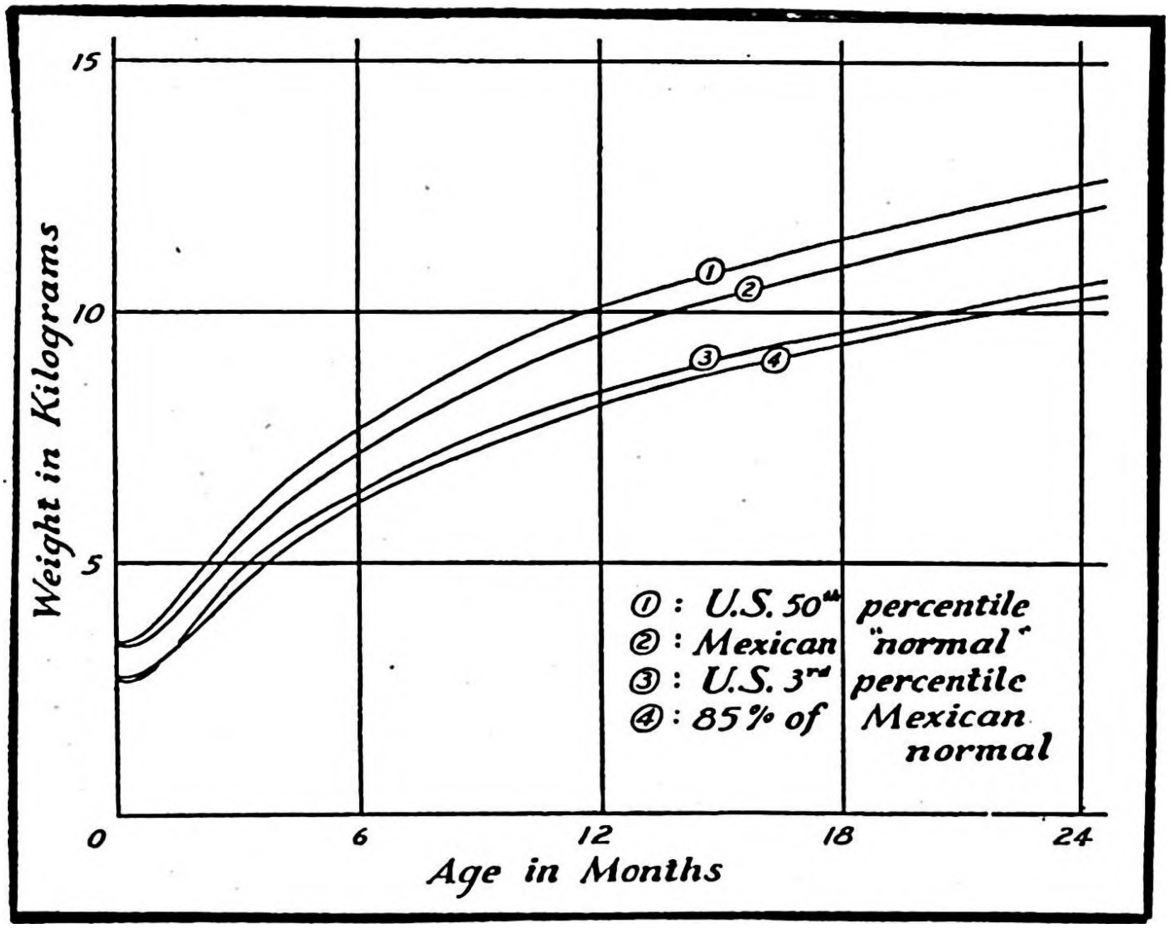

FIGURE 1.

grams, at four months 6,000 grams, at eight months 8 kilograms, at one year 9 kilograms, and at two years 12 kilograms, without significant differences between the sexes. Figure 1 illustrates the comparison of these standards with the norms in the United States.[2] The top

curve (1) shows the 50th percentile of weight according to United States figures. The second curve shows the Mexican so-called "normal." The third indicates the United States 3rd percentile level, and on the lowest graph are plotted weights which are 85% of the Mexican "normal" for a given age.

The average birth weight in Turkey is quite comparable to that in Europe and America,[3] and surveys of Turkish children in several localized studies have suggested that the figures for average children's weights are probably somewhat higher than those in Mexico, but since at present we have no really good statistical evidence of our own, we preferred to use the Mexican standards. This has meant that somewhat fewer children have been rated as malnourished than might otherwise have been the case.

Figure 2 exhibits a sample of infant weights, plotted against age, taken from our own outpatient clinic population. Here we see that 43 percent of all children consulting our hospital under the age of two years were malnourished. The grading used in this study is practically the same as that employed by Federico Gomez of Mexico, except that we have distinguished a fourth group representing the most severe cases. [1]

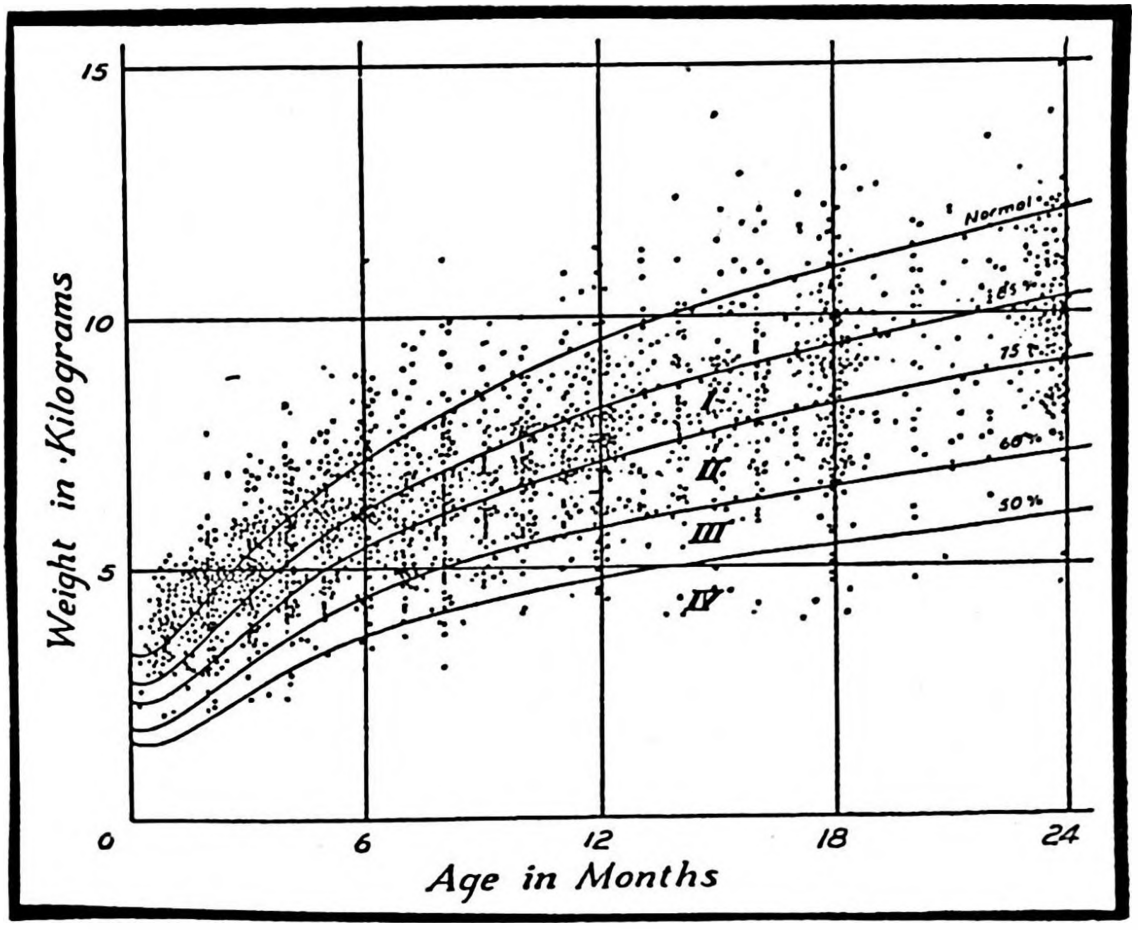

FIGURE 2.

In first degree malnutrition, the patient's actual weight lies between 85 and 75 percent of the theoretical normal for his age. In second degree, it is between 75 and 60 percent of normal. Third degree malnutrition indicates a weight between 60 and 50 percent of normal average, and below 50 percent, the malnutrition is classified as fourth degree. In this graph it is clearly evident that while the majority of our children are comfortably within the "normal" range during the early months of life, large numbers of them fall into the various categories of malnutrition as time progresses. It is logical to assume that this occurs throughout large areas of the world.

How does it happen? Obviously, a great many factors are involved. Ancient cultural patterns and local customs play an important role. It is amazing to see that in most instances the parents and older siblings of a severely malnourished infant may be quite adequately nourished. In many parts of Turkey it is customary to supplement breast feeding with sweetened cereal gruels made without milk, or with very little milk. Whenever the breast milk becomes insufficient, larger amounts of the cereal gruels are given. In the beginning such an infant looks quite happy and satisfied. In many cases the cause of malnutrition is found in the over-long continuation of a diet of gruel, originally introduced to stop diarrhea, and the return of loose stools is regarded as an indication of "sensitivity" to cows' milk. Our villagers do not trust cows' milk, and with good reason: it is, sooner or later, grossly contaminated and pathogenic in its own right. So whenever the breast milk is not available rice water or other starch water mixtures or sweetened tea are resorted to and persisted with. Infants have been brought to our hospital who have had *only* such mixtures for as long as six months. You might wonder how they have survived. So do we.

Acute and chronic infection plays a significant role, but is it cause or effect? Severe malnutrition and infection go hand-in-hand, and we feel that it matters little which comes first —the end result is the same. In certain parts of Turkey and in many unevenly developed areas of the world poverty has to be named as a leading cause of infantile malnutrition. Stresses produced by economic expansion, and limited agricultural practices, are also important.

These factors, and others, produce a situation in which the infant is provided a diet which is grossly deficient in almost every possible way: proteins, minerals, vitamins, even calories. As the child stops growing, then begins to waste away and becomes irritable, he becomes unappealing, if not repulsive, and his care is likely to become deficient as well.

Cardiovascular and genito-urinary anomalies, endocrinological and neurological disturbances, diseases such as fibrocystic disease of the pancreas (mucoviscidosis), probably occur about as frequently in Turkey as in other parts of the world. Yet they do not cause a significant proportion of growth retardation and/or malnutrition.

What are these infants and children like? In the milder degrees they are simply underweight; they may be somewhat short in stature, but otherwise they appear relatively normal. With increasing severity, the weight loss becomes more marked, linear growth slows further, and neuromuscular developmental retardation more noticeable. Without going into elaborate detail about the clinical and laboratory findings, it is sufficient to say that finally they become terribly wasted,with their skin hanging in limp folds, either extremely irritable or apathetic, and, to say the least, they are not difficult to recognize. Our hospitalized patients are practically always very acutely ill because of secondary infection. Interestingly enough, we see relatively few severely malnourished infants who present marked pitting edema and skin changes considered typical of kwashiorkor.

The older students of pediatrics will recognize also that the infants we consider most severely malnourished (third and fourth degrees) would have been considered to be "athreptic" by Parrot, "atrophic" by Czerny, and that according to Finkelstein's classification, they would be said to show "decomposition". Whatever the label, the problem is the same. A few decades ago, infants in these categories were thought to be entirely hopeless cases. Recent reports indicate that anywhere from 35 to 65 percent of them may be saved by proper treatment. A grim prognosis at best. Because of the frequency of these infants in our patient population, we have come to consider them our most important and challenging problem.

I should like to turn now to the problem of the actual management of these infants and children in hospital. The material I will present is derived from our experience in the first year of operation of our hospital. Much is written about these problems, but generalizations are often of little aid in managing a particular case. I hope to get a little beyond the usual generalizations today and share with you some of the specifics we have learned.

In our country, and I am sure Turkey is *not* unique in this respect, the parent rarely brings his infant or child to the doctor and says "My baby is malnourished". Malnutrition develops slowly and insidiously, and often the parent is only vaguely aware that there is a problem. He usually waits until secondary infection

TABLE 1

THE FIRST ELEVEN CASES OF SEVERE MALNUTRITION

Case No.	Age in Months	Admission Weight (grams)	Degree of Mal-nutrion	Main Complication	Days in Hospital	Discharge Weight (grams)	Outcome
1	13	5000	4	Diarrhea, dehydration, rickets, thrush, urinary tract infection, xerophtalmia	43	5030	Unchanged
2	18	4550	4	Dehydration, thrush, otitis media, pneumonia	9	4780	Improved
3	10	5180	3	Diarrhea (E. Coli), dehydration	27	6025	Improved
4	8	3770	4	Dehydration, bacteriemia, thrush	1	—	Expired
5	14	3580	4	Dehydration, bacteriemia	1	—	Expired
6	2y	5910	4	Bronchopneumonia	1	—	Unchanged
7	11	4900	3	Diarrhea, dehydration, otitis media, bronchopneumonia, urinary tract infection	31	5750	Improved
8	6	3600	3	Rickets, cephalhematoma	90	4370	Improved
9	11	4070	4	Diarrhea, bronchopneumonia	1	—	Expired
10	24	7120	4	Diarrhea, rickets, lower respiratory system infection	16	7500	Improved
11	9	4350	3	Diarrhea	1	—	Expired

supervenes, producing symptoms which he recognizes as "illness" as opposed to "weakness" — fever, diarrhea, or coughing, for example — and then brings the child to the doctor. These children, because of their malnutrition, are exceedingly vulnerable to infection and our first task, usually, is to cope with the symptom-producing infection which brings them to us.

In our patients the most frequent serious complications are severe diarrhea, producing dehydration and acidosis, and pneumonia. These problems kill children rapidly and often constitute real emergencies; our house staff is trained to direct primary attention toward dealing with them. Once the child is stable, we can begin to re-nourish him.

What have we lerarned in our first year? Table 1 presents some of the pertinent information about the first eleven severely malnourished infants (third or fourth degree) who were admitted to our hospital in a two-month period during the autumn of 1957. Our results, as you can see, were discouraging, and yet our treatment was in accord with generally accepted pediatric principles. We treated infections vigorously with antibiotics and appropriate supportive therapy, including oxygen. We treated dehydration and acidosis with equal vigor, utilizing 40-60 cc/kg of M/6 sodium lactate, and 30-40 cc/kg of Ringer's solution. Of this total, 30 cc/kg was given rather rapidly ("pushed in") intravenously and the remainder administered by subcutaneous or intraperitoneal infusion. It is worthy of note that in our early months of operation cut-down infusions were infrequent and that we utilized blood only in a limited fashion.

In those children who survived the first few days in hospital —i. e. became stable—we attempted to rely mostly on parenteral fluids until diarrhea ceased, then began oral feedings with exceeding caution. We began with feedings providing 30-40 cal/kg/day and increased these quantities very slowly, in accordance with much of the literature on this subject. In spite of this, our patients showed an exasperating frequency of gastrointestinal disturbances—which the low caloric intake was supposed to prevent — and they gained weight painfully slowly.

How have we changed our management in the course of the past year? Tables 2 and 3 show our overall results on the last 48 infants with third or fourth degree malnutrition admitted to the wards during August and September of this year, comparing them with the first eleven cases, and I must confess some pride in our results. While the two groups are not comparable in numbers, I can assure you that the first

did not unduly exaggerate our mortality and I feel that the current figures accurately reflect the present state of affairs. Of these 48 infants, 10 expired, 2 of them within the first 24 hours, 2 were taken from the hospital by their parents against advice and 3 remained in hospital unimproved. 33 have either recovered or have been in hospital long enough that we can be reasonably confident that they will recover. Many of our malnourished infants who did not present severe complications and in whom vomiting was not striking were not hospitalized; they were sent home on adequate milk formulae and were followed in our outpatient clinic or at their homes. These milder cases with relatively better prognosis are not included in this series.

TABLE 2

OUTCOME IN THE FIRST 11 CASES

Outcome	Number	Percent
Improved	5	45.4
Unchanged	2	18.1
Death	4	36.3
Total	11	100

TABLE 3

OUTCOME IN THE LAST 48 CASES

Outcome	Number	Percent
Improved	33	68.7
Uncertain (still in hospital)	3	6.2
Unchanged	2	4.1
Death	10	20.8
Total	48	100

I mentioned earlier that we treated infection vigorously and were equally vigorous in treating dehydration and acidosis. It soon became apparent to us that vigorous measures must be very gently applied. These infants, when they arrive at the hospital, are in a truly delicate equilibrium and over-vigorous treatment or rough handling is likely to tip the balance in a fatal direction. We have stressed the importance of this in our training programs and now doctors and nurses alike exercise great care in their handling of the children, avoiding unnecessary procedures and deferring others until the infant is better able to tolerate them. This is particularly pertinent in regard

to the management of fluid and electrolyte problems. At present we give all fluids intravenously, by "drip" rather than "push" and at a slow, controlled rate, providing about 150-200 cc/kg for the first full 24 hours. Several things are worth pointing out in this regard: The cardio-vascular status of these infants is such that they seem not to absorb fluids from the extravascular spaces. In fact, we are reasonably certain that in some of our early cases the circulatory status was distinctly aggravated by subcutaneous or intraperitoneal fluids. Hence, we have almost completely abandoned those routes. At the same time, however, we are aware of the dangers of overloading the circulatory system and, as indicated, give the intravenous fluids cautiously and slowly, spreading them over quite a long period.

As regards the type of fluids, we are using less M/6 sodium lactate alone and relying more on a balanced salt solution. We attempt a percentage estimation of deficits, bearing in mind that the appearance of malnourished infants may produce a false impression of the severity of dehydration per se, and usually limit the total fluid volume to 150-200 cc/kg during the first 24 hours. Of this, 50-75 cc/kg is usually lactated Ringer's solution, providing 7-10 mEq/kg of sodium ion and somewhat less chloride. The remaining necessary volume of water is provided as 5% glucose in water. If acidosis is clear-cut and severe, we may initiate intravenous therapy with 20-30 cc/kg of M/6 sodium lactate solution.

Though it is established that malnourished infants are depleted of potassium, we have not seen early evidence of any significant effects of this. However, we have seen typical symptoms of hypokalemia appearing after a few days in hospital. We therefore begin to provide 3-5 mEq/kg of potassium ion, given as potassium chloride, as soon as renal function is well established. It is not ordinarily necessary to continue this for long since the early introduction of milk supplies ample potassium.

The occurrence of hypokalemic symptoms after a few days in hospital when the child was seemingly stable is interesting. Here we suspect the phenomenon is similar to that which occurs in post-acidotic diabetic patients—i. e. serum depletion produced by the rapid shift of potassium ions into the intra-cellular compartment.

Another question is the matter of calcium metabolism. A few of our infants have shown spasms suggestive of hypocalcemic tetany, without other symptoms and with only slightly sub-normal serum calcium levels. Yet they respond well to intravenous calcium. We simply do not have enough data at this point to understand these phenomena.

We utilize blood promptly and freely. Our policy is to limit the quantity at any given time to 5 or 10 cc/kg and achieve larger quantities by frequent administration, giving one or two transfusions daily for several days. We are convinced that the therapeutic effects of blood, improving nutritional status and the level of serum proteins, and enhancing resistance to infection, have made a real difference in the effectiveness of our therapy. We appreciate the fact that such repeated transfusions involve a risk of sensitization — both leukocytes and platelets could be involved. However, we feel that the value in the immediate situation offsets such risks.

Our treatment in the post-acute stage has also changed. Now we no longer consider diarrhea as a contraindication to starting oral feedings. We have fewer relapses, the infants begin to gain more promptly, and the gain, once begun, is more rapid. I might make an illustrative digression. On admission, these infants, regardless of the presence or absence of complications, are apathetic, disinterested in their environment, and often extremely irritable. Early in our experience we were happy within the first month to provoke from one of them a smile, which we consider one of the first indications that the child is really recovering. Now the majority become playfully responsive within the second week, and occasionally we are gratified with a smile in the first week.

This has largely been accomplished, we feel, by early oral feedings, starting as soon as the child will retain them, and using milk primarily. We feel that the exact formula is not of too great consequence; at the moment we rely on diluted milk with 5 percent added sugar. In our situation this is cheap, readily available, and satisfactory. We have learned that fat need not be completely avoided as implied in some of the earlier literature. Another factor which we consider of importance is the caloric intake. Our usual procedure is to start with 50-75 cal/kg and gingerly increase this as rapidly as tolerated. Now we frequently arrive at 150-200 cal/kg, or even more, within the third or fourth week, a level at which many of these infants make dramatic gains. We are able to accomplish this by decreasing the dilution of milk rather rapidly until whole milk is given within a week or so. In addition, we begin supplementary feedings of yoğurt and locally popular milk-starch pudding as soon as we think they will be retained. Both of these foods are rich in minerals and proteins plus calories, and enable us to increase the caloric intake without overextending the volume. Finally, we proceed to pureed vegetables, eggs and meat promptly.

There are several other aspects of care which should be mentioned briefly. We routinely use vitamins in generous maintenance dosage. It is sometimes suggested that these children benefit more from "food vitamins" than "commercial vitamins." We are not sure, but, at a minimum, we feel no harm comes of it. One thing I feel is worthy of mentioning in regard to vitamins. We watch carefully for specific vitamin deficiency diseases in these children and rarely see any of them except rickets. These infants, whose diets are qualitatively and quantitatively deficient in so many respects, seem somehow to fail to respond to individual deficiencies in a typical manner.

In a number of our patients clusters of petechiae were observed, occurring mostly on the lower abdomen. An investigation is being carried out in our hospital into the incidence of purpura, thrombocytopenia and abnormalities of the prothrombin complex in our severely malnourished infants and children. At present we feel that vitamin K should be included in the therapy especially when, for some reason, such as vomiting, milk feeding has to be postponed.

Because of our free use of blood early in treatment and the subsequent early introduction of eggs, we do not find it necessary to use supplemental iron per se.

When, by chance, a severely malnourished infant arrives in hospital without evidence of significant infection, we nevertheless utilize prophylactic antibiotics, feeling that infants with such low resistance deserve extra protection.

There are other interesting facts and I would like to cite a few examples of areas in which we feel we have gained a glimmering of practical insight and which we hope will yield helpful information. We have seen some infants who were in a semi-comatose, shock-like state and undoubtedly near death. They did not respond readily either to blood or to electrolyte solutions. We tried giving rather rapid injections of 10 percent glucose, wondering if they might be hypoglycemic. The response in a few cases bordered on the dramatic. Since our initial empirical trials, we have been able to obtain blood samples for measurement of serum glucose levels in a few cases and found the glucose ranging from 5 to 10 mgm. per cent. What does this mean? It is logical to consider it related to tissue depletion of glycogen and we are seeking to learn more about it. At the moment, we can not define a syndrome—the symptoms are not those typical of acute hypoglycaemia — nor explain the findings, but we are sure that the quick administration of glucose has been life-saving in a few instances.

This discussion would not be complete without stressing the obvious importance of nursing care. For good medical care during the acute stage, close cooperation between doctors and nurses is essential. Later on, the simple mechanics of feeding these infants is time-comsuming and requires patience, and gentle, loving nursing care.

Nor should I neglect to mention the importance of parent education prior to the time infants are finally discharged. Unless the home environment is improved, the conditions which produced the malnutrition initially are likely to persist and produce a recurrence. We encourage our house staff to give the parents careful, understandable instructions regarding the type of food, and its preparation, needed by their child. We try to convey to parents that, for their children, good food is the best medicine, and explain to them what good food means.

What of our therapeutic failures? Is there an irreducible minimum mortality in severe malnutrition? Undoubtedly there is, for practical purposes, but we do not pretend we have achieved it. Pediatricians the world around are called upon to try to resurrect moribund infants and children —from whatever cause. We have our share of these among malnourished infants. Certainly there is a stage in the pathophysiology of severe infectious diseases beyond which threre is no recall. Some cells undoubtedly "die" before others and simply cannot revive.

We would prefer to ignore this possibility, however, and operate on the more optimistic assumption that, with adequate knowledge and understanding, if the child is alive on admission, we will be able to arrest immediately the downhill progress and gradually restore normalcy. Some of these infants die for reasons not at all clear, and gaining the understanding necessary is a real and fascinating challenge.

It is an interesting and gratifying experience for our medical and nursing staff to follow these severely malnourished infants in the wards and watch them improve day by day. There is no monotony because in no two cases is the treatment exactly the same, nor is the response identical. Individualized management is ever important.

The treatment of malnutrition is one of the most expensive and time-consuming processes in medicine. In countries where malnutrition remains a major pediatric problem there is never enough room in hospital for all such infants needing care. The cost of providing personnel and adequate facilities to meet these needs on a worldwide scale staggers the imagination. With our present facilities and devoted, able staff we may be able to salvage even more than 75 percent of these children, but what does this mean when we consider that we serve only a small fraction of the total affected population of our country?

The obvious answer is that prevention is the key to the solution of the problem. Malnutrition, after all, is preventable. But this is not easily achieved either. Unfortunately, prevention of malnutrition is not simply a matter of lining children up and giving them an injection. It *would* be nice if there were such a thing, providing a year's supply of vitamins, minerals and proteins in one shot, wouldn't it? That day has yet to arrive, however, so we must grapple as best we can with the multitude of factors mentioned earlier: cultural, social, environmental and economic. We know that, if prevention is the key to the problem, education is the key to prevention. Countries like ours can take heart from the fact that countries like yours have made such striking progress in the last five decades.

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